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THE BRITISH JOURNAL
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FEBRUARY, 1943

COMMUNICATIONS

A THIRD ENTITY OF PRIMARY SYMMETRICAL FATTY CORNEAL DYSTROPHY

BY
J. A. CONWAY *and* A. LOEWENSTEIN
GLASGOW

WE have thought fit to place on record an account of a case of symmetrical lipoidosis corneae, and with it certain cognate observations. We are moved to do this for two reasons. One is that we consider this case to be unique of its type. A second reason is to express the hope that this contribution may stimulate interest in and enquiry and research into a condition, which is probably not too uncommon and about which, apparently, our knowledge is still very incomplete.

Before describing the case, certain general facts regarding fatty change in the cornea, might usefully be recalled.

The young, normal, human corneal tissue is—as far as is known—fat free. Fatty compounds are found only in senile and pathological corneal tissue. The breakdown of protein into fat has so far never been observed as having taken place in the cornea, although we would not rule out this possibility. Our assumption is that visible fat found within corneal tissue, is fat derived from the blood, and that it originates from ingested food. According to the histological researches the nourishing plasma leaves the vascular network at the limbus, and its fat is oxydised in the corneal lamellae. Possibly if too much fat enters the corneal tissue, or if the normal oxydising power of the cornea is not strong enough, or if both

conditions collaborate, the fat is stored, and not oxydised. It might be mentioned here that on the other hand cases have been observed with a high cholesterol content (about 1000 mgm. per cent., the normal being 150-200 mgm. per cent.), this feature being of long duration, and yet without the appearance of a fatty corneal deposit.

A lipoidal corneal arc, however, can be produced in the rabbit by feeding it on cholesterol in olive oil. This arc is very similar, but not quite identical, with the senile arc. Sometimes the eye of a rabbit fed in this way shows in addition to the arc a diffuse fatty dystrophy (sklerokeratitis lipoides, Loewenstein, 1942).

It will be recalled also that fatty changes are observed in corneal tissue of reduced vitality. Kerato-malacia is an instance, another being the fatty infiltration found in eyes debilitated by scars or inflammation. These changes are explained as being due to defective *local* oxydising power of the cornea.

The study of fatty changes in the cornea has taught us to distinguish between *primary* and *secondary* dystrophies. In a primary fatty dystrophy we witness the appearance of fat in a cornea which careful clinical examination by all the methods at our disposal, reveals to be otherwise normal. In a secondary dystrophy signs of disease or injury in the eye are observed to account for the appearance of the visible fat. In cases of primary symmetrical fatty corneal dystrophy both eyes are impaired, not inflamed, the corneal epithelium is undisturbed, the parenchyma is usually more or less diffusely opaque with distinct greyish-white spots. In some cases the spots coalesce to form a homogeneous yellowish white mass. No vascularisation is observed or secondarily only, due to a foreign body reaction. Twelve cases are known in the literature. No successful line of treatment is published. Histologically one of us found slight fatty changes in Bowman's membrane and minute fatty droplets in the anterior corneal lamellae. The middle layers are entirely destroyed and full of fatty masses and cholesterol crystals, while tiny fatty droplets occur in the posterior lamellae. Descemet's membrane is fatty in its anterior third only. No sign of inflammation was discovered. Aetiology of this appearance is obscure.

A second primary symmetrical fatty corneal dystrophy exists in dysostosis multiplex (Gargoylism or G. Hurler's syndrome). Twenty-five cases have been described so far: in twenty-four cases the very uniform corneal changes have been recognised. Corneal surface is smooth, grey-white, minute opacities are distributed all over the cornea throughout the whole parenchyma. Some of the patches are somewhat larger. No other changes in the eyes have been observed. Histologically a fatty infiltration is observed within the spaces between the corneal fibres. Lipoidal substances are found in Hurler's disease in liver, spleen and lymph nodes. A linkage to Tay-Sachs's amaurotic idiocy is worth considering as certain

cerebral changes described in Hurler's disease are reminiscent of those in amaurotic idiocy.

Up to date these two groups of primary, symmetrical lipoidal dystrophy are known. We add here a new form of this mysterious corneal change.

History

M. M., female, single, age 35 years, came for eye examination, with the complaint of difficulty in vision for near work. She was well-built, but of delicate appearance, and there was an obvious right lobe goitre swelling. Unaided superficial examination of the eyes showed a generalised "cloudiness" of both corneae. Her family history revealed nothing that assisted in forming an estimate as to the probable cause of her eye condition. She has indoor work in an office. The patient herself had been in good health until about twenty years of age. when she began to have kidney trouble. Later on she noticed the thyroid swelling. Shortly before coming for eye examination she had had a long period in bed with kidney disease. She stated that she felt far from well, and was tired and breathless. Her general symptoms indicated failing cardiac and kidney competence.

Bulbi both slightly exophthalmic in an equal degree. Lid fissures 12 mm. looking straight forward. Four pingueculae of considerable size and of sulphur-like colour. Normal eye movements. It is remarkable that the red blood corpuscles within the conjunctival vessels move regularly for the most part, but, to the observing eye,

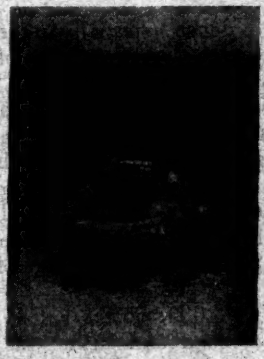

FIG. 1.

Primary symmetrical nephrotic corneal lipoidal dystrophy.

they disappear in some apparently empty vessels. Here the movement is visible again in bends only.

A broad lipoidal arch of 1 mm.—1·3 mm. in diameter does not reach the limbus (Fig. 1). The lucid interval between arch and sclera is slightly hazy. The arcus lipoid can be dissolved at the narrow slit and high power into coarse grey granules and fades towards the centre of the cornea. The peripheral margin of the arc (Fig. 2), is very irregular and reminds one of the ice lumps of the cinguliform dystrophy. The whole cornea is equally infiltrated by very small greyish-white dots. They stretch from the subepithelial level (Fig. 3) throughout the whole thickness of the

FIG. 2.

Irregular "lucid interval" between lipoidal arc and sclera.

FIG. 3.

Slit-lamp narrow beam. Dots in Bowman's M. thicker.

corneal lamellae and are dense, especially in the region of Bowman's membrane. They cover the whole extent of the cornea equally without producing a bulge in the epithelium. There is no epithelial defect. There are no corneal vessels, and no corneal nerves were discovered. No changes on the posterior surface of the cornea were present, nor any corpuscular elements in the anterior chamber. Iris is of brownish-yellow colour and normal structure. Pupil mobile and active. Lens clear. Fundi reveal typical Gunn's dots, especially on the nasal side of the disc. Broad and hard reflexes of the retinal arteries. Some peripheral twigs of the retinal veins are of unequal calibre.

Vision, R.E.: 6/5 (?) +3.0 D.=6/5. L.E. 6/6 +3.0 D.=6/5.

The examination of blood and urine was done by Dr. James Eaton, in charge of the biochemical department of the Victoria Infirmary, Glasgow, to whom we are very much indebted.

Blood: urea: 54 mg. per cent. Plasma cholesterol: 216 mg. per

cent. Plasma total fat: 885 mg. per cent. Plasma albumen: 1·65 per cent. Plasma globulin: 2·02 per cent. Albumen/globulin ratio: 0·816.

Therefore the blood urea is high. Cholesterol is slightly increased. Albumen is very low and globulin high. The albumen/globulin ratio is very low. There is albumen in urine. According to Dr. James Eaton the chemistry of the blood corresponds to that which would be expected in a nephrotic type of renal lesion, but the cholesterol is not so high as is usually found.

Unfortunately not all the tests desirable and indicated could be carried out due to the unwillingness of the patient to allow the investigations to proceed any further.

Conclusion

We consider this to be an undescribed entity. We would place it in the group of "feeding" fatty dystrophies, in the sense of a corneal over supply of fat from the bloodstream. It is not a cholesteraemia, but a lipaemia. A general disturbance in fat metabolism, or even a local disturbance cannot be ruled out. The first sign is the arcus lipoides. A lipoidal arc is found in young people, in cases of nephrosis with high blood fat figures, as one of us has described. The peripheral corneal tissue acts as a filter to protect the optically important central corneal areas. In our cases this filter broke down, and the central part became flooded with fatty droplets.

The increased blood fat alone cannot cause the fatty dystrophy, as the highest degree of lipaemia in diabetes, with tremendous fat masses in the blood serum does not provoke, as far as we know, the fatty corneal changes. We assume that it is the *long lasting* lipaemia which allows the fatty infiltration. There is a second supposition possible, namely, that a lower local corneal metabolism, a deficiency in its oxydising power, arises from altered endocrine influence. Admittedly we have no proof for this supposition except for the struma and the exophthalmos. No basal metabolism was done due to the lack of collaboration on the part of the patient.

We would call attention to the strange kind of corneal lipoidal arc. Its peripheral margin is like broken ice, sub-epithelial. Fractured plates correspond to calcareous incrustation in Bowman's membrane, and remind us of cinguliform corneal degeneration. The lucid interval is very irregular.

We formed the impression that corneal metabolism was severely damaged by an unknown cause. It is not impossible that it was due to the continuous over-flooding of the peripheral corneal tissue with fat rich plasma. The establishment of fatty infiltrates within the peripheral corneal fibres causes wear and tear of this tissue, and

renders difficult or impossible the proper filtering action of this part of the cornea.

One of us has observed this remarkable clinical picture once only. We are convinced other less obvious cases have been over-looked. In the future we shall be on the look out for cases of this type, and now that attention has been drawn to it, perhaps others will report similar cases. A suggested name for this affection might be "primary symmetrical interstitial fatty corneal dystrophy with lipoidal arc, due to nephrotic lipaemia." Further investigations by clinicians and biochemists will be required.

A CASE OF MUSTARD GAS KERATITIS UNDER CONSTANT OBSERVATION FOR A PERIOD OF TWENTY YEARS

BY

T. L. DE COURCY.

LIVERPOOL

IN a recent article in your journal by Miss Mann and Dr. Pullinger the authors expressed regret that we had few details of the treatment given to the earlier cases of delayed mustard gas keratitis. As few ophthalmologists can have had the opportunity of keeping in close touch with a case of this dread disease for a period of over twenty years, a few observations on its course, the treatment given —harmful or otherwise—and the gradual adaptation of the different therapeutic aids as they became available, may be of interest. Possibly the deductions drawn, though of no scientific value, may be helpful as to what to avoid and what to make use of in the treatment of any cases which may still develop even after so long a period.

Even now there is much to learn about delayed mustard gas keratitis—the reason for varying periods before onset of ulceration; why, under similar circumstances of infection "one should be taken and the other left"; whether focal or general disease may be a factor predisposing the gassed candidate to ulceration; in what measure the concentration of gas affects the issue. Most important of all, will the study of this form of keratitis become mainly academic owing to the stoppage of supply, or are we to visualize the future treatment of a vast throng of patients of both sexes and of all ages?

My patient, F.W.S., is now a man, aged 62 years, still an imposing figure, six feet two or three inches in height and proportionately built. Before the world war he was a County tennis

player and an outdoor man in every sense. He had an excellent war record, winning the M.C. and before the war ended was second in command of his regiment. What is more he is a man of great intelligence, holding an important business position, and this intelligence he has used throughout his treatment in noting any fresh symptoms or any harmful reaction to treatment, and in his wholehearted co-operation which has led to any measure of success achieved up to the present.

It was his reaction to games that induced him to consult me in the early summer of 1922. He " could not stand the light as well as he used to; he could not follow the flight of a golf ball or judge the distance to the hole; he had just been beaten 6-0 6-0 in a tennis tournament by a man to whom he used to give points." He " knew that in the early forties he could not expect to play games as well as formerly, but why this sudden deterioration within the last month or two "?

In May 1918 a gas shell had burst at a distance of thirty or forty yards when " at rest " in the back area, and he thought some of the actual fluid entered his eyes. He described the usual symptoms of acute gas poisoning—treated at Base and afterwards English Hospitals for some months. In August 1918 he was passed by a Medical Board and again sent out to France, but he asked the Board to make a note that his sight was not so good as before.

On demobilization he repeated that he was still not seeing so acutely and was again sent to a Medical Board. He states that his early reception was distinctly unfriendly till the ophthalmologist on the Board had made his examination, after which the atmosphere improved. He was then informed that everyone who had been gassed was making a claim regarding eyes, but that he was evidently one of the very few who had really suffered and was granted a 15 per cent. pension.

My examination revealed slight scarring of the corneae just below the pupil (rather more marked in the right eye), a small degree of hypermetropic astigmatism and corrected vision of 6/12 6/6, but no visible acute condition apart from intolerance to light.

In my opinion the process of disintegration of the corneae had then started with these subjective symptoms, as during the next three years I had to change his glasses at intervals of less than a year owing to increasing astigmatism which gradually assumed the mixed variety. For example, early in 1925 his refraction was :—

$$\text{R. } \frac{+0{\cdot}75 \text{ sph.}}{-3{\cdot}0 \text{ cyl. } 10^{\circ}} = 6/18 \qquad \text{L. } \frac{+0{\cdot}75 \text{ sph.}}{-2{\cdot}5 \text{ cyl. hor.}} = 6/6$$

His accommodation was also poorer than one expected.

It was unfortunate that during this period the slit-lamp was not part of the armament of the ordinary ophthalmic surgeon, but later on with its advent, I was able to take a more intelligent interest in my patient.

At this period, however, there was no evident vascularization of the corneae and no slit-lamp to detect the formation of crystals as described in Miss Mann's article. It was only on the evidence of the rapid changes in refraction, indicating degeneration of the corneae, that later on one felt certain that the pre-ulcerative phase of gas keratitis had started when the patient first consulted me. It must be remembered that during this stage the prospect of future ulceration was not in my mind as there were at that time very few such cases described in this country.

The next stage in my patient's corneal history began late in 1925 when on his honeymoon in the Riviera (he was also a late starter in matrimony) the first attack of ulceration started in his right eye causing his precipitate return home. The left eye was not attacked until 1926 and he never exceeded a period of more than a year without an attack in one or the other eye till 1937, since which date no serious ulceration has taken place.

Each attack lasted from a few weeks to three months varying in severity, each leaving behind fresh corneal destruction which was gradually encroaching on to the pupil areas. The ulcers were not large but generally multiple and involving the deeper corneal layers. The rugged scar areas left after each attack reduced the vision in the right eye so much that I ceased to try to correct it by glasses in 1928. Till 1938 he relied chiefly on his left eye and reached the stage in which he could only see to read with glasses when the book was held close to the eye.

It is only necessary to mention two or three other clinical points. In 1924, towards the end of the pre-ulcerative stage, it was noted that both corneae were anaesthetic. It may be the practice of some ophthalmic surgeons to test the sensitivity of the cornea of all their patients coming for refraction, but after all life is short. My patient had made no complaint of any inconvenience to suggest such a condition, and it was only the repeated refractive changes which induced me to explore further the state of the corneae. It would now be interesting to know if the corneae had been anaesthetic since 1918.

Two very subtle sources of danger were exposed—that of the possibility of injury by foreign bodies, and still more (in this particular case) from the friction of occasional ingrowing lashes. This latter source was the cause of several attacks of ulceration, which have been less frequent since the patient has attended regularly, every month or so, to have any suspicious lashes removed.

Crystals in the substance of the corneae were noted soon after the first slit-lamp examination—in the late 1920's. Cholesterin crystals were then plentiful and long needle-like clear crystals were also seen in abundance. Latterly cholesterin deposits have been absent or very rare.

Blood vessels encroaching on the cornea were present even in the earlier stages, but not in leashes—mainly single vessels which in the earlier stages did not give much trouble. It would be interesting now to have had more precise notes as to their appearance and their distribution in the cornea after crossing the limbus, as a comparison with the cases now being seen. What were noted as "bullae" were of frequent occurrence on the surface of the corneae.

Later and particularly in the last two years three small "islets" of blood vessels horizontally across the centre of the pupil have interfered with the vision of the left eye. Their treatment will be referred to later.

Treatment.—Not appreciating at first that gassed corneae should not be treated in any way differently from ordinary corneal ulceration, the usual methods were applied and the first two attacks were probably prolonged for that reason. It was soon apparent that carbolizing the ulcers did definite harm and that such drugs as atropine, dionine, quinine and zinc applied in anything like their usual strength acted as a source of irritation. I referred my patient to Sir William Lister who induced me to curb my over enthusiastic treatment, and in preaching the gospel of masterly inactivity—at any rate as regards drugs—helped me considerably in the treatment of this case. It is interesting to note that sixteen or seventeen years later Miss Mann hints at the same doctrine in the article previously referred to. Weak solutions of the above drugs and many others were made use of and the usual antiseptics applied, but Locke's solution has been the sheet anchor in his local treatment. Cod liver oil and parolein were also helpful. Probably had saline and oily solutions been used alone, which would have needed some courage at that time, the result would have been equally effective.

On three or four occasions I performed tarsorrhaphy of the Wheeler type, once involving a period of one year in the left eye, twice for at least six months in the right. During the occlusion of the eye he had no attacks of ulceration, but after the lids were opened ulceration took place fairly soon. The results of tarsorrhaphy were on the whole disappointing, and in the light of present methods probably would be unnecessary.

X-rays were used therapeutically in 1936, as by this time the state of the corneae was causing grave anxiety. On the whole X-ray treatment may be said to have cut short the ulcerative

periods and certainly did not irritate the eyes. At a stage when the battle appeared to be going against us it was a relief to find any treatment which enabled the eyes to hold their own, but X-ray must be applied by an experienced radiologist.

Whatever the eventual outcome may be of this hardly fought battle there was a decided stand in 1937 and since then a period of renewed hope resulting in improved business and social life. It came about through news of a friend of the patient's whose corneal ulceration had greatly benefited by the application of short-wave diathermy under the direction of Jess of Leipzig. We decided it was worth a trial and this decision proved a very wise one, as a lengthy spell of treatment gave results that one could scarcely have hoped for. The electric heater had been used for years with benefit, but this localized heat from the short-wave diathermy, repeated afterwards at Moorfields, and now available in Liverpool, should it be needed again, holds prospects of success far ahead of anything previously tried in this patient's case. There has been no further attack of severe ulceration since diathermy was first started, and that at a period when things were going from bad to worse.

For some years my own feeling had been that it would be worth while to try the effect of contact lenses, to improve the much impaired vision and also to shield the cornea, but could not get anyone to agree that it was worth the risk. My patient returned from Germany with " stock " but reasonably well fitting contact lenses which he was able to wear for short periods. I was about to send him to Budapest for fitted lenses when Dr. Dallos came to London, and the lenses supplied by him have proved an enormous success. They can be worn for ten hours or so at a time and though occasionally when irritation of the eyes occurs one or other lens has to be left off for a few days, the general improvement in sight has been miraculous. The right eye which had been almost discarded as a visual organ has enabled him to conduct his business when the left eye was for one reason or another temporarilly out of action, and with the left eye plus contact lens he has recently had 6/9 vision and been able to read newspaper type comfortably.

Incidentally the only other case of mustard gas keratitis which has come under my care, recently developed a severe hypopyon ulcer in his only remaining eye. A large part of the cornea, including the pupil area, became infiltrated with pus and vision was reduced to perception of light. A lengthy period of short-wave diathermy at the Eye and Ear Infirmary, Liverpool, resulted in a healed but badly scarred cornea, when the odds seemed all in favour of panophthalmitis. Applications to the Ministry of Pensions for the fitting of a contact lens resulted in his removal

to London where fitting took place at Moorfields and an iridectomy was performed with very satisfactory results.

It is conceivable that the saline contained in the contact lenses is beneficial to the corneae and may be the only treatment required. However difficult it may be to rid our minds of the routine treatment for corneal ulceration I feel convinced that the best treatment for gassed corneae would be cod liver oil drops and saline, together with diathermy and contact lenses, but this opinion is based on the experience of only two cases. Other factors have of course, to be considered and in the case of F.W.S. the recent encroaching of blood vessels on the corneae and the " islets " of vessels over the pupil area were causing concern. On the advice of Miss Mann it was decided to cauterize these vessels at the limbus. As any operative procedure had to be performed under the aegis of the Ministry of Pensions (his pension now being at the 80 per cent. rate) the patient was recently operated on by Mr. Humphrey Neame at Moorfields, who, partly by cautery and partly by the diathermy point cut off a great number of vessels at the limbus, and the islets have practically disappeared—an excellent result. There is no reason why this procedure should not be repeated if necessary in the future, though the reaction has been very marked and one eye remained red for a considerable period.

Conclusions

The opportunity of being in close touch with a case of delayed mustard gas keratitis for twenty years must be shared by few ophthalmologists and for this reason it may be worth putting on record the clinical features most worthy of note and the main lines of treatment—good and bad—which were tried out, and the vicissitudes of fortune during that period.

Any measure of success attained has been due to the patient's keen intelligence in noticing any abnormal symptom and reporting it at once (I encouraged the use of the telephone), and to his loyal support in every fresh line of treatment, together with the helpful advice and assistance given by many surgeons whose interest was aroused in the patient. What is also very noteworthy, and gives some idea of the character of the patient, is the fact that never once have I heard him bewail his fate.

Risking some inevitable repetition the following points in this case appear to me most noteworthy.

1. To judge from the scarring of the corneae when the patient was first seen, actual ulceration must have taken place at the time of gassing (1918).

2. Subjective symptoms appeared four years later (1922) with photophobia and report of "bad judgment" in visual efforts.

3. Rapid changes in refraction in the "pre-ulcerative" period of those years (1922-1925) in which degenerative changes presumably took place in the corneae to account for these variations in refraction.

4. Anaesthesia of the corneae first noted in 1924 but probably present since the initial attack. Is anaesthesia always present in delayed gassed corneae?

5. The necessity for constant observations of this particular case in order to remove aberrant lashes and watch for foreign matter which might injure the anaesthetic corneae.

6. The onset of the ulcerative stage seven years after his initial gassing, and its recurrence at intervals during the next twelve years. The moderate vascularization accompanying these attacks.

7. The deposit of cholesterin and other crystals in the corneae in the earlier stages of the disease and their gradual disappearance.

8. Drugs used as drops had to be prepared in much weaker solutions than for ordinary keratitis. The suggestion is made that oily drops, normal saline, or Locke's solution may have been the only applications necessary for this case in which iritis was never a prominent feature. Pure carbolic must never be used.

9. The disappointing results of tarsorrhaphy, as soon after reopening the lids ulcerations again took place.

10. The possible good results of X-rays carefully applied.

11. The undoubted benefit of short-wave diathermy.

12. The protection provided to the corneae by fitted contact lenses and the striking increase of visual acuity resulting from their use.

In this connection there should be a word of warning. The actual fitting of the lenses was a very severe strain on the patient and caused slight ulceration. Fitting had to be suspended for some three weeks while the corneae recovered.

13. The late appearance of "islets" of blood over the pupil areas and their disappearance after peritomy.

14. The fact that no focus of infection was found in the patient to predispose him to mustard gas keratitis—the usual tests having been made at the time of his first attack of ulceration.

15. Lastly that after twenty adventurous years the patient leads a practically normal social and business life.

HAEMANGIOMA OF THE ORBIT, REMOVED BY OPERATION

BY

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SPECIALIST IN PATHOLOGY

MAJOR E. F. KING, R.A.M.C.
SPECIALIST IN OPHTHALMOLOGY

and

MAJOR H. W. RODGERS, R.A.M.C.
SPECIALIST IN SURGERY

PRIVATE B, aged 30, a regular soldier, reported sick in Hong Kong in February, 1940.

The man gave a history that eighteen months previously the right eye became inflamed and felt " tense " and that he experienced a sense of numbness in the right temple; these symptoms passed off in a few days. For the last six months he had experienced intermittent attacks of neuralgic pain behind and over the eye, lasting for several hours; these were increasing in severity and leading to some loss of sleep. At no time had he noticed any head noises.

He was seen by an ophthalmic specialist who reported a mild degree of right proptosis. General and special examinations failed to reveal any other abnormality. He was kept under observation and as the proptosis slowly progressed he was returned to the United Kingdom.

When seen in this country in January 1942 he showed well-marked right proptosis, directly forwards, unassociated with any retraction or lagging of the upper lid. No pulsation or bruit was detected. The movements of the globe were surprisingly full though there was some limitation of its peripheral excursions, particularly upwards and inwards. No sensory changes were detected in the cornea or in the skin adjacent to the eye. Deep palpation of the orbit failed to reveal any mass. The pupil reacted well to light and on accommodation. The optic disc and surrounding fundus were normal. There was slight, though definite, nasal contraction of the peripheral visual field (see Chart—Fig. 1). The blind spot was normal and no central scotoma was detected.

Vision $= \frac{6 \text{ - } +1{\cdot}00 \text{ sphere}}{18 \text{ c } +1{\cdot}00 \text{ cylinder } 180^\circ} = \frac{6}{9}$ and J.1. The man had never worn glasses and was emphatic that the vision in the eye had deteriorated in the last two years and it seemed possible that the hypermetropia and astigmatism were, in part, artificially produced by pressure on the posterior pole of the eye.

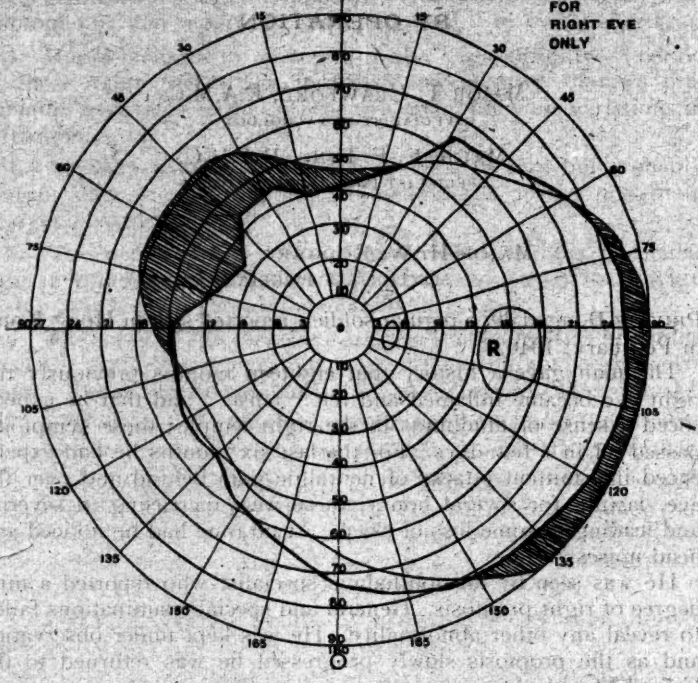


FIG. 1.

The left eye was essentially normal, and the vision $-\frac{+0{\cdot}50 \text{ sph.}}{+0{\cdot}50/180^{\circ}} = \frac{6}{5}$.

X-rays showed both optic canals of equal size and there were no abnormal shadows in the orbits or sinuses.

Complete general and special investigations were carried out, particularly in regard to a primary malignant tumour elsewhere in the body or any changes in the paranasal sinuses. All were negative.

The diagnosis of a slowly growing tumour of the orbit within the cone of muscles was made and surgical exploration advised.

Operation

The usual approach to the retro-ocular contents by reflection of the lateral orbital wall was used. The alternative transcranial exposure, by removing the posterior two-thirds of the orbital roof,

was not undertaken in this case as it was thought improbable that the orbital tumour had any intracranial connections.

Several incisions in the bone have been recommended. Of these the widest exposure, recommended by Kocher, is given by dividing the bone along the following lines (see Fig. 2): (A) From the zygomatic process of the frontal bone to the middle of the inferior orbital fissure. (B) From the lower and outer angle of the orbit,

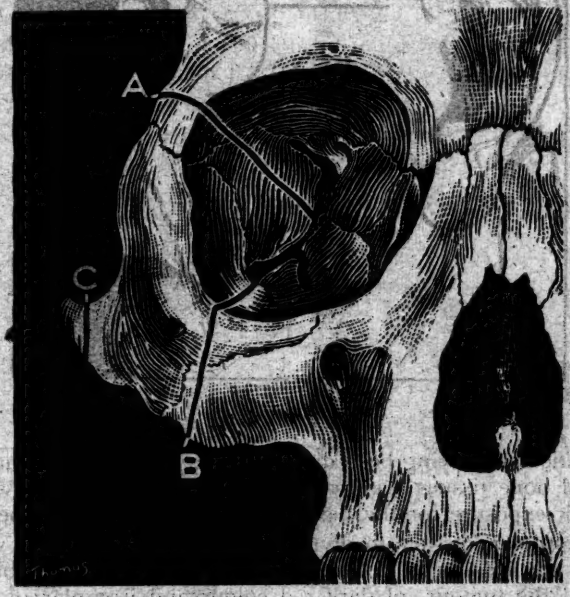


FIG. 2.

backwards and inwards to the anterior extremity of the inferior orbital fissure. (C) Across the zygomatico-temporal suture.

The preliminary skin incision (see Fig. 3) was carried deeply to the zygomatic process of the frontal bone. The orbital contents were displaced medially by the cautious separation of the thin and ill-defined orbital periosteum until the orbital fissure was reached. In this a malleable retractor was placed to protect the orbital contents and, keeping in close contact with the calvarium, the first (A) of the bone incisions described above was begun with a small saw and completed with a chisel. Before the next cut (B) was made a blunt hook was placed in the anterior end of the inferior orbital fissure to act as a guide, and the incision was made sufficiently lateral to avoid the maxillary air sinus. Subsequently, the

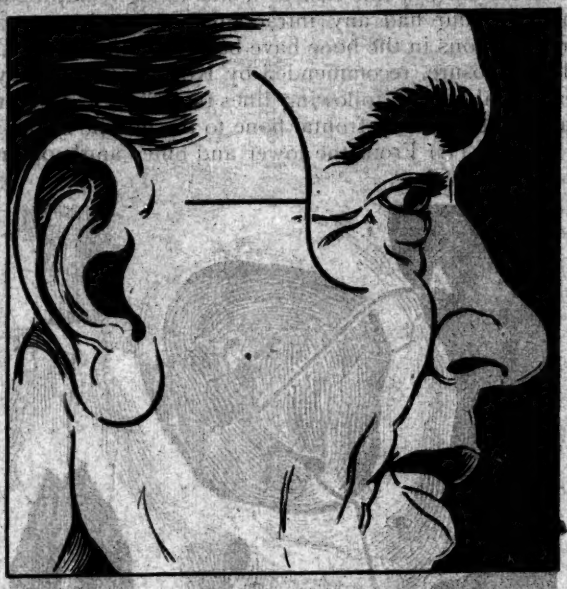


FIG. 3.

zygomatic arch having been divided, the zygoma with its attached soft and bone tissues was readily displaced backwards giving an excellent exposure for the next important stage of the operation.

It was not necessary to divide the lateral rectus which was retracted upwards by a hook. Blunt dissection of the orbital fat revealed the tumour, of dark red colour and irregular in shape. It was lying within the cone of muscles in close apposition, but not attached, to the optic nerve. The tumour was readily and completely removed by separation with the forefinger.

This operation destroyed both branches of the zygomatic nerve. A more conservative approach might have preserved the zygomatico-facial branch at the expense of a more cramped field of operation with its attendant risk of irreparable damage to the delicate retro-ocular structures.

The only difficulty experienced in closing the wound was the accurate resetting of the orbital margin; but good alignment was finally attained by a few strong catgut sutures.

There were two notable features of this wide and apparently mutilating exposure, namely, the absence of haemorrhage, and the small degree of permanent disfigurement produced.

Convalescence following the operation was uneventful apart from marked oedema of the upper lid which, on examination six weeks later, had subsided considerably.

The proptosis was entirely relieved and movements of the globe were full apart from some weakness of the external rectus muscle. The pupil, which previous to the operation had been normal, was now semi-dilated and reacted sluggishly to light and on accommodation. There was also marked cycloplegia.

The vision was $\frac{6}{24}$ c +2·50 sph. $= \frac{6}{6}$, and it was necessary to add a +2·0 sph. to obtain J.1.

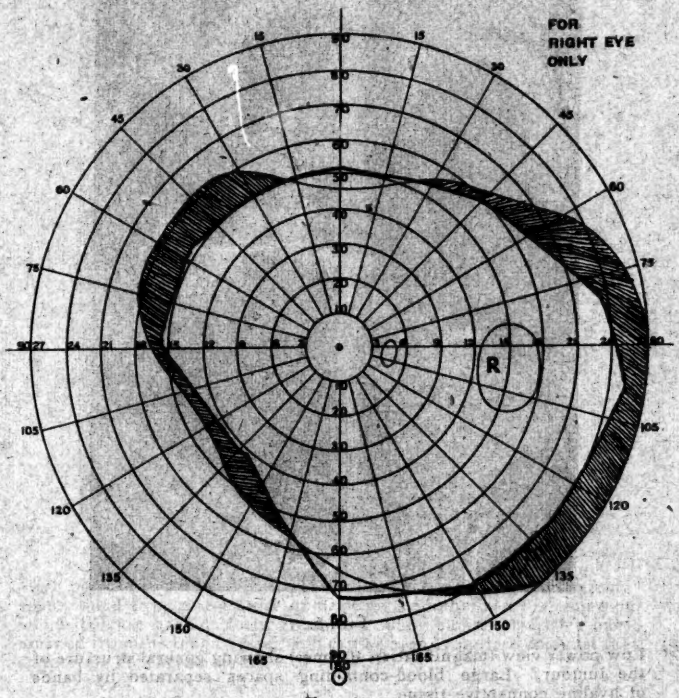


FIG. 4.

It seems that this post-operative increase in hypermetropia was due to the cycloplegia which made manifest the whole of the latent hypermetropia.

The fundus was still normal. The visual field was now full there being no trace of the nasal contraction previously noted (see Chart—Fig. 4).

Pathological Report (See Figs. 5 and 6)

The tumour is roughly egg-shaped and measures 2·3 cm. in length by 1·5 cm. in width. The cut surface has a fleshy appearance with areas suggesting blood clot.

Microscopic examination shows the tumour to be composed largely of cavernous blood-containing spaces lined by well-developed vascular endothelium, separated by fibrous tissue of

FIG. 5.

Low power view (magnification 40 times) showing general structure of the tumour. Large blood-containing spaces separated by bands of hyaline connective tissue.

varying density. In addition to the cavernous spaces a few small arterioles with muscular walls are present. While the bulk of the tumour is formed of these mature vascular elements there are, in addition, areas in which solid cords of endothelial cells separate primitive capillary channels. These areas show numerous mitotic figures and clearly constitute the growing foci of the tumour.

Numerous blood cells have escaped into the interstitial tissue between the cavernous spaces, and haemosiderin granules are present, in large numbers, within the cytoplasm of endothelial cells and macrophages. At the edges of the tumour the vascular elements

FIG. 6.

High power view (magnification 250 times) showing a blood-containing space, lined by a single layer of flattened endothelial cells, some of which contain haemosiderin granules. Many blood corpuscles have escaped from the blood vessel, and can be seen lying free amongst the supporting connective tissue.

fade gradually into the " capsule " formed of a loose fibrous and adipose tissue.

The tumour is clearly a mixed capillary and cavernous haemangioma arising, presumably, from the blood vessels lying amongst the orbital fatty tissues. The possibility of its origin from the sheath of Schwalbe has been considered, but there is no histological evidence to support this.

While the tumour is essentially simple in nature, the microscopic appearances suggest the possibility of rapid growth or rapid recurrence following incomplete removal.

Conclusion

It is generally agreed that haemangioma, particularly the cavernous type, is among the commonest of the orbital tumours, and that its complete removal is usually practicable. Points of note in this case were the early numbness and neuralgic pain around the eye, unaccompanied by any sensory changes, and the evidence of pressure on the optic nerve, reflected in loss of the nasal peripheral visual field, though the optic disc was normal on ophthalmoscopic examination.

Injury to the nerves supplying the iris, ciliary body, and lateral rectus, was an unfortunate sequel to the operation, though probably this was unavoidable in the mobilisation of a tumour of this size in so confined a space.

OCULAR COMPLICATIONS IN RELAPSING FEVER*

BY

MAJOR J. BRUCE HAMILTON

A.A.M.C. 2ND A.I.F. ABROAD

Introduction

WHEN Mackenzie in 1830 wrote his "Practical Treatise on Diseases of the Eye," he mentioned that ocular complications occur and should be watched for in relapsing fever. In those pre-Pasteurian days no idea of the aetiology was possible, nor was it anticipated that numerous types of relapsing fever, each due to a different spirochaete, were to be revealed.

Mackenzie's description was of the cosmopolitan louse-borne type of the disease. The delayed ocular complication of amaurosis followed later by acute iritis, as described by him, is typical of the complications I shall enumerate. Also he noted carefully that the iritis recovered long before the amaurosis cleared. He saw in the epidemic in Glasgow 36 cases of ocular complication within 3 months, which is a much greater number than I have examined in the 12 months May 1941 to May 1942.

Since Mackenzie's day several cases of choroiditis accompanying relapsing fever have been reported in the literature; but it is very

* Received October 20, 1942.

doubtful if they were cases either of louse-borne or of tick-borne spirochaetal relapsing fever as we now know it. After the discovery of the various spirochaetes many writers, chiefly from Teutonic clinics, commented on the ocular complications of this disease; but so far as I am able to ascertain (and it is difficult in a theatre of war to be sure on this point), no further detailed reports have been made since the end of the last war.

The European spirochaete was the first to be differentiated and it was described by Obermeier of Berlin in 1873. Since then six further types of spirochaetes have been discovered, each causing the same relapsing type of fever with definite modifications of symptomatology. Later in this paper I shall briefly sketch the parasitology of relapsing fever, and it will be then seen that a still clearer differentiation of the various spirochaetes is very necessary. This clearer differentiation is urgently needed, for it is becoming very apparent that the complications of one are not the complications of the other. It may also explain why, in 92 cases seen at the 7th Australian General Hospital between May 5, 1941 and May 5, 1942 of which 28 cases came from the Western Desert and Libya, and 63 came from Syria, only four cases of iridocyclitis were seen, all from the Western Desert and Libya. This fact will be stressed further.

Ocular Complications

Elliot (1920) and Feigenbaum (1940) mention the following ocular sequelae of relapsing fever:—

1. Oedema of the lids.
2. Iridocyclitis with vitreous opacities.
3. Optic neuritis.

But in my limited experience of the disease I have found only fibrinous iridocylitis with gross vitreous exudate of alarming proportions which may manifest itself in two forms:—

(*a*) Acute iridocyclitis with gross vitreous exudate (Cases 1-3).

(*b*) Chronic cyclitis with gross vitreous exudate accompanied by persistent headache (Case 4).

Both these types have an excellent prognosis and appear to run approximately the same prolonged course. It could be expected that choroiditis would be revealed in all affected eyes when the vitreous has cleared; but I have not found that so, nor have any nodules been found on the iris. The posterior synechiae in the former type have been very gross; but these have ruptured with

ease after mydricain injections. Quite often the exudate into the vitreous is so marked that the fundus picture is screened from view by a dense veil; but even this clears with time, leaving a normal fundus with normal vision.

Those who are sufficiently interested in this subject to have read Mackenzie's account of the Glasgow epidemic will remember that despite the subsidence of all inflammatory signs in the eyes he found that the amaurosis (undoubtedly due to the gross vitreous exudate) persisted for a considerably longer period.

The case records which are to follow will give an intimate picture of what the clinician should be prepared to find when the human eye is attacked by a spirochaete of the relapsing fever group.

Treatment

Unlike syphilitic uveitis the prognosis of relapsing fever uveitis as seen in the Middle East seems to be extremely good, and should not cause the oculist any definite concern. Heat and mydriatics are all that are required. No one has been impressed by the efficacy of the salvarsan group in affecting the course of the disease and the fact that one patient with iridocyclitis (Case No. 1), after his fourth injection of N.A.B., had a fever relapse on the following day with a temperature of 103°F. bears out this fact. What I am still asking physicians is, "can a patient have malaria and relapsing fever at the same time, and if so will the pyrexia of the former diminish the number of relapses of the latter?" I have had no satisfactory answer to date. We know malaria therapy influences the course of an infection of spirochaeta pallida; but will it similarly influence these distressing recurrences of Mediterranean relapsing fever?

Major Jones, of the Trans-Jordan Frontier Force, has recently given me his treatment for the relapsing fever found in the Amirate of Trans-Jordan. It is as follows:—

1. 3 c.c. Bisoxyl (B.D.H.) (bismuth oxychloride) intramuscularly every 5 to 7 days. Three injections.
2. Tablets of Stovarsol (4 grains): 4 to 6 tablets daily in courses of three days starting on the day of injection.
3. Mistura potassium iodide, and 500 mgm. daily of ascorbic acid, and 60000 international units of vitamin A daily are given during fever.
4. 100-200 mgm. of ascorbic acid, and 30000 international units of vitamin A are given when each patient is afebrile.

I have had no opportunity of testing the efficacy of his treatment on the Syrian or Libyan fevers; but I thank him for handing the

information on to me. He has found the treatment most successful, and seldom sees more than one relapse as a result.

Concurrent Complications

Here I refer to several concurrent complications which have arisen during the treatment of the nine cases recorded in this paper. They are: (*a*) Facial paralysis. (*b*) Herpes febrilis. (*c*) Encephalitis.

(*a*) Facial paralysis. I do not want to create the opinion that the 7th cranial nerve is the only one which may be involved; but it is the only affected one which I have encountered. I hear from other authorities that the 6th nerve also has been found involved. I also want to stress this fact, (which again has not come under my direct notice) that in some cases the facial paralysis becomes permanent. But I have no knowledge that keratitis e lagophthalmo has ever arisen as a consequence.

(*b*) Herpes Febrilis. The virus of this complaint has produced so many troublesome keratides in the Middle East following almost any pyrexial attack, that it is only to be expected that odd cases of herpetic or dendritic keratitis will occur after repeated attacks of relapsing fever. Although I have seen many such cases following malaria, sandfly fever and coryza, I have only seen one case following relapsing fever (Case 3). It must be remembered that this patient had had 3 intramuscular injections of milk, so that the keratitis may have resulted from this therapy. Such cases must be watched for, however, and if they occur it should be remembered that the virus of herpes febrilis only awaits the high pyrexia precipitated by another disease to multiply and attack the victim about the lips, maxilla, lids or cornea. I hope to record these observations in a later paper (*see* p. 80).

(*c*) Encephalitis. Incessant headache due to a chronic encephalitis is a very common accompanying feature of spirochaetal iridocyclitis; but it too responds to rest and patience, and should not be confused with more serious cerebral lesions of a pyaemic or a neoplastic type. (Cases 6 to 9).

Parasitology

Professor S. Adler of Jerusalem has been good enough to help me with this section by loaning me unpublished articles from his Department at the Hebrew University. I also must acknowledge reference to Hewlitt & McIntosh (1932) Stitt, Clough & Clough (1938) and Manson (1940).

Manson (1940) has tabulated the seven known spirochaetes of relapsing fever as follows:—

Spirochaete	Site	Natural Transmitters
1. Recurrentis or Obermeieri or Novyi ...	N. and S. America ... / *N. and W. Africa* ... / North China ... ... / Europe ... ... / India	Lice
2. Persica ...	Iran ... ... / N.W. India	Ticks
3. Sogdianum ...	Palestine	Ticks
4. Hispanica ...	South Spain ... ... / *Morocco*	Ticks
5. *Duttoni* ...	Central Africa ...	Ticks
6. Turicatae ...	California	Ticks
7. Venezuelensis	Central and S. America	Ticks

A.I.F. Technical Instructions No. 16 designates the Palestine and Syria spirochaete as Sogdianum, and the Libyan and Western Desert spirochaete as Hispanica. Adler refers to the Syrian and Palestine organism as Spirochaeta Persica. It is thus obvious that there is some lack of uniformity in the nomenclature.

But let me turn to the figures of cases from the two regions east and west of the Nile admitted to the 7th Australian General Hospital.

Cases from the Western Desert—28. Of these four developed iridocyclitis.

Cases from Syria—63. Of these none developed iridocyclitis.

Cases from Greece and Crete—1.

We know conclusively that the Syrian and Palestinian disease is carried by ticks found in the many caves especially about Sidon. We are not yet clear as to the name of the spirochaete; but whatever be its true name it does not appear to produce iridocyclitis with any readiness.

On the other hand, the natural transmitter of the Western Desert spirochaete is very doubtful, and further the type of spirochaete seems very uncertain. We know from the century-old figures of Mackenzie that the louse-borne fever is very prone to ocular complication. He saw 36 cases in three months in Glasgow in 1843. During the years 1843 and 1844, out of 1,877 eye admissions to the

Glasgow Infirmary, no less than 261 cases suffered from ophthalmitis following relapsing fever.

The guinea-pig test has been held specific for the tick type; but Adler says "whereas infectivity for guinea-pigs is a practical proof that the spirochaete concerned is not louse-borne, the reverse is not of universal application." Further, he quotes Nicholls and Anderson who have suggested that in North Africa the same spirochaete may be carried by either louse or tick. This last remark may throw light on the confusion of spirochaete and vector in the Western Desert, and at least allow for my theory (gleaned alone from clinical findings) that the Western Desert fever is louse-borne and the Syrian is tick-borne. If this is so it explains the above quoted disparity in ocular complications from the two groups.

Diagnosis

As the condition of iridocyclitis often follows the last relapse of fever it has been difficult, even with the thick drop blood method or in the cerebrospinal fluid, to discover the spirochaete in the film of some of my patients. In two of the four patients with iridocyclitis the spirochaete was never found.

The following two methods have recently been suggested to me; but have not so far been given an adequate test.

1. If the organism cannot be located in the blood or the cerebrospinal fluid, the spirochaete may sometimes be recovered from the bone marrow following sternal puncture.

2. Even more successful is the following: A small quantity of blood is run into a little citrate solution. The specimen is then centrifuged and separates into three layers. A tiny, fine pointed pipette is used to remove material from the central "white" layer, and this is examined for spirochaetes.

Case Records

1-3	Acute Iridocyclitis.
4	Chronic Cyclitis and Encephalitis.
5	Facial Paralysis.
6-9	Encephalitis with Headache.

CASE 1. Male, aged 23 years

April 4, 1941. Admitted to 8th British General Hospital: Pyrexia, general aches, scattered rhonchi left chest. Had been in Western Desert. Pulv. Doveri gr. V and Aspirin gr. X nocte, Mist. Expectorant t.d.s.

April 13, 1941. Generalised aches and pains, plus temperature.

April 16, 1941. M & B 693 tabs. 2 t.d.s. Temperature normal. Improved.

April 18, 1941. Transferred to 9th British General Hospital. Admitted as Coryza. Temp 98·4. Recovered. No further treatment required.

April 22, 1941. Transferred and admitted to 2nd Australian General Hospital. Feels well but weak. Nothing abnormal detected.

April 25, 1941. Temp. elevated. Spirochaete recovered from blood.

May 6, 1941. Slight relapse.

May 29, 1941. No further relapse.

June 4, 1941. Admitted to 7th Australian General Hospital. Has had trouble R.E. for few days. Went to 1st Aust. Conv. Depot one week ago, after relapsing fever. R.E. became painful with poor vision next day. Following day could hardly see at all from R.E.

Examination:—Injected ++. Cells on posterior corneal surface. Post synechiae ++. Pigment on anterior lens capsule. Good reflex. No fundus details. R.V.=<6/60, <J.12. L.E.= Normal. L.V.=6/5, J.1.

R.E., Unguentum Atropine and Cocaine 2 per cent. Steaming.

June 5, 1941. Temp. 103°F.

June 6, 1941. Pupil fully dilated. N.A.B. 0·45 gm. Temp. 103°F.

June 8, 1941. Temp. 99°F.

June 10, 1941. Pupil dilated. Much vitreous opacity. Optic disc not clearly seen N.A.B. 0·6 gm. Temp. 99°F.

June 11, 1941. Fundus seen through vitreous exudate seems very pale. No active retinitis seen.

June 14, 1941. N.A.B. 0·6 gm.

June 18, 1941. N.A.B. 0·6 gm.

June 19, 1941. Vitreous much clearer. Temp. 103°F.

June 20, 1941. N.A.B. 0·6 gm.

June 21, 1941. Temp. 98°F.

June 25, 1941. R.V. 6/9, L.V. 6/5. Fundus of right eye appears normal.

June 28, 1941. Cease steaming. Continue gut. atropine 1 per cent. thrice daily.

July 3, 1941. R.V. with +0·75 D.sph.=6/6 pt. Add +4·0 D.sph.=J.1. L.V. 6/5: J.1. R.E. Old k.p. Pupil fully dilated. Vitreous opacities clearer. Fundus normal. L.E. No k.p. Optic disc is normal.

July 4, 1941. Discharged to 1st Aust. Conv. Depot.

CASE 2. Male, aged 37 years.

October 16, 1941. Left eye has been bloodshot for three days. Pain in head and eyes for the past two months. Came on during relapsing fever. Was in Tobruk. No spirochaetes found in blood film while in 4th Aust. Gen. Hospital on September 7, 1941, but has had 3 attacks of fever since August 1941.

Examination:—R.V. 6/6, J.1 at 16". L.V. 6/36, J.12 at 6". R.E. Normal. L.E. Injected ++ Cornea bright. Pupil active to light. New vessels on iris. Optic disc not seen (undilated).

Atropine 1 per cent. left: Pupil dilates irregularly with posterior synechiae nasally. Optic disc normal; but fundus not clearly seen owing to vitreous opacities.

Treatment. Ung. atropine and cocaine 2 per cent. and steaming left eye.

October 18, 1941. Left subconjunctival mydricain, 5 minims.

October 20, 1941. Pupil fully dilated.

October 24, 1941. 240 lymphocytes but no spirochaetes in cerebospinal fluid. No spirochaetes in blood. Temp. 101°F.

October 27, 1941 Temp. normal.

November 3, 1941. Kline negative.

November 5, 1941. Refraction left eye under atropine: with +0·75 D.sph. +0·25 D.cyl. ax. 45°=6/6. Fundus normal.

November 10, 1941. Discharged.

CASE 3. Male, aged 38 years.

April 27, 1941. M.R.S. (1) Sacro-iliac strain. (2) Pyrexia of unknown origin.

May 16, 1941. At 2nd A.G.H. Fibrositis.

September 27, 1941. Fever, followed by two relapses in November and December. Patient had been at Tobruk.

February 6, 1941. Admitted to 7th Australian General Hospital. Left eye has been sore for a week. Scum over pupil.

Examination: R.E. Normal. L.E. Injected ++. Pupil fully dilated. Tension normal. Much pigment on anterior lens capsule. Much vitreous haze and floaters. Fundus appears normal. R.V. 6/5, J.1. L.V. 6/60.

February 8, 1941. Blood film for relapsing fever negative. Treatment, atropine 1 per cent. and steaming. Mist. sod. sal. Milk injections.

February 4, 1942. R.V. 6/6 J.1. L.V. 6/60. Not improved. Left eye shows gross vitreous opacities, but is white.

March 4, 1942. R.V. 6/6. L.V. with +4·0 D.cyl. ax 150° =6/18.

March 17, 1942. Increase in vitreous floaters. One tooth removed.

March 30, 1942. Left eye red. Marginal keratitis. Vitreous clearer. Treated with silver nitrate 2 per cent. paintings for three alternate days.

April 8, 1942. Cease atropine. Eye excellent.

April 13, 1942. Left eye with −2·50 D.sph. +3·0 D.cyl. 75° = 6/18 pt., J.2 at 12". Cornea clear. Pupil contracted. Vitreous clearer. Fundus appears normal.

April 19, 1942. Left eye red, multiple corneal erosion + + + silver nitrate 2 per cent. painting for 3 successive days.

April 25, 1942. Patient discharged to 6th Australian General Hospital.

CASE 4. Male, aged 39 years

October 21, 1941 to January 17, 1942. Initial fever on day of admission to 2nd Australian General Hospital. Had come from Tobruk. During fever had severe headaches and was relieved by lumbar puncture. On October 28, 1941, cerebospinal fluid pressure was 240 mm.; cells 170 leucocytes per cmm., mainly lymphocytes were found increased to 70 mgm. per c.c. Spirochaetes were found in blood smear during fever on October 22, 1941; but no type mentioned. Had four relapses on November 6, 28, December 5 and 16.

February 3, 1942. Admitted to 7th Australian General Hospital. No fever for one month. Last five days has had a scum over right eye. No trouble otherwise. Never wore glasses.

Examination: R.E. White ring on cornea. Cells on posterior corneal surface. Gross vitreous opacities but no fundus lesion seen. Pupil dilates regularly. L.E. Normal. R.V. 6/9 J.1. L.V. 6/6 J.1.

February 11, 1942. Increased vitreous floaters.

February 17, 1942. Headaches severe.

March 3, 1942. R.V. with +0·75 D.cyl. ax. 90° = 6/5. L.V. = 6/5.

March 11; 1942. Right vitreous opacities still marked. Both fundi normal.

CASE 5. Male, aged 25 years

September 15, 1941. Admitted to 1st Australian General Hospital with relapsing fever contracted at Tobruk. Persistent headaches followed. Facial paralysis started October 7, 1941. Has had five relapses; last one January 1, 1942. Spirochaetes found in blood.

March 7, 1942. Admitted to 7th Australian General Hospital. Headaches unchanged, but facial paralysis is cured.

Examination: R.V. 6/5, J.1. M.H. 0·50. L.V. 6/5, J.1. M.H. 0·50. Maddox rod: 0-0. Maddox wing: 4-6 exo. -0. R.E. Normal orbicularis. Good corneal sensation. Eye normal. L.E. Weak orbicularis. Poor corneal sensation. Eye normal.

Refraction under 2 H. and C.: R.E. with +0·50 D.sph./+0·25 D.cyl. 80° = 6/5. L.E. with +0·50 D.sph. = 6/5.

Comment:—No ocular cause for headaches which are undoubtedly due to relapsing fever encephalitis. No treatment for facial paralysis indicated.

CASE 6. Male, aged 23 years

February 16, 1942. No injuries prior to enlistment. Stretcher bearer. Contracted relapsing fever in Tobruk. Has had five attacks since September 1, 1941. Last attack one month ago. Pains at back of both eyes for past month; but no relation to reading. Spirochaetes found in blood.

Examination: R.V. 6/5 and J.1. L.V. 6/5 and J.1.

No muscle imbalance. Convergence normal. Under homatropine and cocaine: R.V. with +0·50 D.sph. = 6/5. L.V. with +0·50 D.sph. = 6/5.

Right fundus shows several small patches of old retinitis and one large recent patch below. Left fundus normal.

Comment:—This patient has an active retinitis possibly due to relapsing fever; but not typical. Kline test and rest advised.

March 17, 1941. Kline test was negative. X-ray of eye negative. Retinitis is unchanged and is certainly not due to relapsing fever. Headache due to encephalitis.

CASE 7. Male, aged 32 years

January 22, 1942. An out-patient. Present general health good. Contracted relapsing fever in Syria. Had about eight relapses. Since then eyes ache and gets headaches after reading or close work. Has never worn glasses. Spirochaetes in blood.

R.V. = 6/5 and J.1. L.V. = 6/5 and J.1.

Dilated, right fundus shows in temporal periphery a small patch of retinitis with corresponding vitreous exudate. May be a sign of relapsing fever; but needs no further treatment.

January 26, 1942. After refraction under homatropine and cocaine, and post mydriatic test, prescription given for constant wear as follows:—R. +0·25 D.sph./+0·50 D.cyl. 90° = 6/5 and J.1. L. +0·25 D.sph./+0·50 D.cyl. 90° = 6/5 and J.1.

It is very doubtful if these headaches are due to a refractive error—most possibly they are due to encephalitis following the spirochaetal infection.

CASE 8. Male, aged 24 years

March 30, 1942. Bad headaches since contracting relapsing fever in Syria, extending from June to December 1941. Print blurs on reading. Had 12 relapses. Spirochaetes found in blood.

Examination: R.V. 6/6 and J.1. L.V. 6/6 and J.1.

Maddox rod: 4 exophoria −0. Maddox wing: 6 exophoria −0. Rather poor voluntary convergence. Fundi normal.

Refraction:—Right, with +0·50 D.sph./+0·25 D.cyl. ax. 175° =6/5. Left, with +0·50 D.sph./+0·25 D.cyl. 50°=6/5.

Constant glasses ordered to relieve headaches which, however, are primarily due to the relapsing fever encephalitis.

CASE 9. Male, aged 25 years

April 8, 1942. Contracted relapsing fever at Tobruk in July 1941. Spirochaetes isolated. Had four attacks. No eye complications but severe headaches ever since.

Examination: R.V.=6/9 and J.1. L.V.=6/6 and J.1.

Maddox rod: 0−0; Maddox wing: 4 exophoria −0. Poor convergence. Gross old anterior retinitis in right and left fundi. No vitreous opacities. Retinitis most probably the result of boxing rather than due to relapsing fever.

April 13, 1942. Correction after refraction and post mydriatic. test: Right, with +0·25 D.cyl. ax. 75°=6/5 most J.1. Left, with +0·25 D.sph.=6/5 and J.1.

Spectacles not required. Fields full each eye to 5/333 white. Headaches due to chronic encephalitis after relapsing fever, and accentuated by a convergence insufficiency. For orthoptics.

April 15, 1942. Kline negative. Headaches showing marked improvement with convergence stimulation.

Comment on Nine Cases

These cases I think illustrate several points:—

1. That although we had 92 patients suffering from relapsing fever at 7th Australian General Hospital, every one whom I saw with iridocyclitis contracted his fever in Tobruk (Libya) or the Western Desert.

2. Although there were four cases of iridocyclitis, all having very similar features, in only two of these was the spirochaete isolated. When ocular complications arise, the febrile period has usually (but not always) passed. In the afebrile period it is often impossible to find the spirochaete even in the cerebrospinal fluid or thick film method. I have no doubt that all four cases were of the same clinical entity.

3. The four patients with uveitis had had respectively 5, 3, 3 and 5 relapses before the cyclitis appeared, giving an average of four attacks before onset.

4. Two of the four patients with uveitis had relapses following the onset of iridocyclitis. One had two such attacks and the other had one.

Case	Attacks before iridocyclitis		Relapses after
1	5		2
2	3		1
3	3		—
4	5		—
Four cases averaging	4	and	0·75

5. In this small series all cases were unilateral, although Elliot (1920) quotes one bilateral case from literature.

6. Although two cases showed retinitis, in both cases this was inactive and I am certain had no relation to the spirochaetal fever.

7. No case of optic neuritis was seen although 92 cases of relapsing fever passed through the hospital in the year.

8. One patient with facial paralysis came under observation; but he is recovering. This is not always the result, and in at least one of our 92 cases permanent bilateral facial paralysis resulted.

9. Headaches are a very worrying complication both with and without ocular complications of iridocyclitis. The iridocyclitis produces little pain; but the encephalitis is persistently aggravating. When it occurs without apparent ocular disease, the patient is often referred for a refraction opinion as illustrated in several of my cases.

10. The percentage of iridocyclitis following relapsing fever is difficult to determine; but possibly it is in the vicinity of 20 per cent. from Western Desert cases and nil from Syria. As the complication is a delayed one the affected persons are often treated in one hospital for the initial disease, and in a second one for the iridocyclitis.

11. I have no helpful suggestions regarding the prevention of ocular complications, or of lessening the severity of the attack; but in my experience the visual result is nearly always satisfactory. The lack of response of our patients to intravenous injections of N.A.B. is disappointing and we must look to our research workers and pharmaceutical chemists for help in this direction.

Acknowledgement

In conclusion, I thank many members of the staff of the 7th Australian General Hospital both medical and clerical for their help and advice in treating these cases and preparing these notes. Also I acknowledge my gratitude to our Commanding Officer for permission to publish these case reports.

REFERENCES

1. ADLER, S. (1942).—Personal Communication.
2. A.I.F. Instruction No. 15, 1941
3. CASTELLANI, A. and CHALMERS, A. J. (1913).—Manual of Tropical Medicine, p. 925. London.
4. DUKE-ELDER, W. S. (1940).—Text Book of Ophthalmology, p. 2151, Pt. III. London.
5. ELLIOT, R. H. (1920).—Tropical Ophthalmology, p. 505. London.
6. HEWLETT, T. R. and MCINTOSH, J. (1932).—A Manual of Bacteriology, p. 442. London.
7. MACKENZIE, W. (1830).—Practical Treatise on Diseases of the Eye.
8. MANSON, P. (1940).—Tropical Diseases, p. 211. London.
9. RIDLEY, F. and SORSBY, A. (1940).—Modern Trends in Ophthalmology, p. 48. London.
10. STITT, E. R., CLOUGH, P. W. and CLOUGH, M. C. (1938).—Practical Bacteriology, p. 140.

NOTES ON FORMS OF KERATITIS PRESUMABLY DUE TO THE VIRUS OF HERPES SIMPLEX

BY

MAJOR J. BRUCE HAMILTON

A.A.M.C.—ABROAD

Introduction

THE idea of writing these notes has been germinating for some considerable time but it has been brought to the surface by many facts and figures, both written and verbally communicated to me recently. Besides these intimations I have been able to make certain personal observations myself, in the Middle East, on this particular subject and these should be noted immediately.

In the *Brit. Jl. of Ophthal.* of January, 1941, I recorded a survey of superficial punctate keratitis in Tasmania over an 8½ years period. I here clearly showed from recent clinical and laboratory observations that this disease, at least, is due to the virus of herpes simplex. At the same time I indicated that superficial punctate keratitis in man was accompanied by a number of other corneal lesions, but these two facts do not yet seem to have entirely grasped the attention of my readers, nor have other important facts in my paper been appreciated. One has only to

turn to the *Arch. of Ophthal.*, of February, 1942, and read the separate reports of Gifford and of Davis, to realise that the position, even in the U.S.A., could be clarified with advantage. The distinction between a bacterial and virus keratitis seems, in many minds, to be almost indistinguishable, and I feel this difficulty is due to the fact that a 50-year-old conception, that all external ocular infections are bacterial in origin, has taken too long to die. Further, little attempt has been made seriously to culture the conjunctiva from the virus standpoint and, when a bacterial culture is reported on as " no growth," further cultural attempts are not made to distinguish the organism. Personally, I feel that in the majority of cases a bacterial keratitis can be distinguished clinically from the virus type with ease, and I should like to detail the distinguishing points forthwith.

When is a Keratitis due to the Virus of Herpes Simplex?

I think a virus simplex keratitis has several cardinal clinical features, which I intend again to (Hamilton, 1941) repeat and possibly enlarge upon. They are as follows :—

First, this particular type of infection often appears following a true or artificial pyrexia. That is, it may follow a pyrexia of malaria, relapsing fever, influenza or coryza, or an artificial pyrexia following T.A.B. injection. This last point has been abundantly confirmed by me in the A.I.F., and other writers have recorded attacks of herpes labialis after T.A.B. inoculations. Further, I feel even inclined to say that a sudden change from an arctic to a tropical climate is sufficient to precipitate an attack—undoubtedly the Polish Divisions, recently moved from the Russian winter to the Palestine summer, have been sorely afflicted by the disease. Gordon (1941) confirms this climatic influence. At least, marked changes in body or atmospheric temperature can be recorded in the majority of cases.

Secondly, the relapsing nature of this keratitis has impressed itself on everyone, both in Australia and in the Middle East. Not only are relapses in one eye common but also a spread to the second eye (at any time during or after the attack in the primary eye), is not an unusual feature. The short immunity to the virus of herpes simplex is recorded by Mackie & McCartney (1938). I, personally, doubt whether this particular virus produces any immunity whatsoever.

Thirdly, the total absence of acute iritis and the small amount of conjunctival discharge are both interesting features. I never use atropine for virus keratitis, except the disciform type, and have not found my judgment misplaced.

Fourthly, the protean nature of the lesions of the cornea, which may range from multiple corneal erosions to disciform keratitis (see Fig. 1). The most usual forms, however, are a superficial punctate keratitis, marginal keratitis or dendritic ulcer. Davis (1942), in the United States of America, has recently reported a superficial punctate parenchymatous keratitis, and I have lately seen a member of the Voluntary Aid Detachment with a "kaleidoscopic" keratitis varying from day to day in its configurement, commencing as a lattice form and ending with multiple erosions. I find that the canoe-shaped lesions of marginal keratitis are the most confusing to many ophthalmologists, but they respond rapidly to lid applications of silver nitrate 2 per cent.

Fifthly, lesions of the lid are not uncommon. The frequency of lid lesions I pointed out in 1941, and I attempted to explain this by suggesting that virus keratitis is a neurotrophic lesion of the ophthalmic branch of the trigeminal nerve. These skin lesions may be in the form of petechiae, vesicles or warts. Mackie & McCartney (1938) consider the virus of herpes simplex is both neurotropic and dermotropic. Since residing in the Middle East, I have been impressed with the frequency with which seborrhoeic dermatitis and pyoderma have accompanied a virus keratitis, and have pointed out this more than coincidental happening to our consulting Dermatologist; so possibly the virus is both neurotropic and dermotropic.

Sixthly, as is only to be expected, cultures of the conjunctival sac invariably show "no growth" or normal saprophites. I have never seen a conjunctival culture reveal any helpful evidence in a virus keratitis of the types at present being described, nor have I ever been able to persuade a bacteriologist to culture the conjunctival secretion for this particular virus.

Seventhly, if treated on correct lines the recovery of this particular keratitis is usually rapid and the visual results excellent. Recurrences may occur but are less likely if the treatment to be enumerated below is followed implicitly.

How may these Eyes be Treated

If it is primarily realised that we are really dealing with a herpes simplex (or febrilis) infection of the eye, analogous to recurring herpes febrilis (or recurrens) of the lip, we shall have burnt down the first barrier in our path, and if we appreciate that these herpetic lesions of the lip are best treated by applications of methylated spirits or tinctura iodi mitis and not by hot applications of any sort, we are over our second barrier.

Thirdly, the rationale of omitting heat is obvious once we realise that it is pyrexia which is the main precipitating factor

of all herpes simplex lesions. So, to omit local heat in any form is of the most paramount importance in treating this type of keratitis. On the other hand, ice compresses, I know, are definitely helpful and valuable.

Fourthly, having decided that local heat is inadvisable, why aggravate the condition by artificial pyrexia such as T.A.B. intravenously or milk intramuscularly? They too are strongly contra-indicated in all types of keratitis of simplex origin.

I recently saw a patient who lost the sight of his right eye following repeated T.A.B. injections for marginal keratitis. Six weeks later his left eye developed a dendritic ulcer and one application of carbolic cured that without visual loss. From this case alone it is obvious that it is not generally realised that marginal keratitis and dendritic ulcer are the same infection in different frockings. Who would think of giving T.A.B. intravenously for dendritic ulcer, so why exhibit it for marginal keratitis?

Fifthly, having discarded heat, let me next discard atropine because, with the possible exception of disciform keratitis, I never use atropine in the disease under review and have never regretted its absence. Herpes simplex virus does not cause iritis. If this observation is universally appreciated the present scarcity of atropine sulphate will be greatly relieved.

Sixthly, auto-inoculation is not practical in keratitis unless there is a vesicular skin lesion as well. Even then, I have never attempted it as I have found that simpler means of aborting an attack are at our disposal.

Seventhly, we have found that spirit or tinctura iodi mitis are efficacious in "drying up" the skin and lip lesions of herpes febrilis, and it appeared to me that a similar method would be applicable to the ocular lesions. We have, for several decades, applied pure acid carbol. to all dendritic ulcers with pronounced success, so is it unreasonable to apply a similar astringent to the other manifestation of the same disease, such as marginal keratitis or superficial punctate keratitis? Spirit did not seem indicated and tincture iodi mitis appeared to cause undue pain, I, therefore, tried an old and very dependable ocular astringent, namely, silver nitrate in 2 per cent. solution, but not 1 per cent. solution as is so often used. At first I applied it only to the palpebral conjunctiva of the upper and lower lids, and the result was dramatic. Later, I became bolder and applied it to the corneal lesions themselves, but I usually leave this last adventure for the more advanced cases. The applications are made at the first inspection, then again 36 hours later, and after that at slightly longer intervals till the cornea is healed and the eye is white. When the eye is definitely white I make an extra and final application which I find frequently prevents a recurrence. This treatment is almost

unfailing in superficial keratitis and marginal keratitis but I find it of little use in multiple corneal erosions. For this I reserve four-hourly instillation of guttae zinc sulphate and adren. chloride: grains one of zinc sulphate to the ounce of water.

For the dendritic ulcers I find nothing superior to applications of pure acid carbol. on a tooth pick, while for disciform keratitis I do not feel we have any reliable treatment except rest and patience.

The Technique of Silver Nitrate Application

I must, before leaving the application of silver nitrate, make several pertinent observations :—

(*a*) The eye should be well cocainised and adrenalised before the application of 2 per cent. silver nitrate. I usually make three instillations of cocaine 4 per cent. and two instillations of adrenaline chloride (1·4000) before I attempt the painting. These instillations are carried out at two minute intervals and, while I have never seen this full local anaesthetization produce any deleterious effect, it certainly maintains the patient's confidence.

(*b*) I also never wash out the eye immediately after the painting, and even if the whole corneal epithelium develops a "milky" hue, this is no case for alarm and irrigation. The cornea will rapidly regain its normal lustre.

(*c*) After the painting I usually instil, on a glass rod, unguentum butyn & metaphen (Abbott) into the lower fornix. This procedure maintains a mild anaesthesia of the cornea and conjunctiva for an hour or more, and allows the pain of the conjunctival ischaemia to pass off relatively unnoticed.

(*d*) The eye should never be wrapped with pad and bandage but protected only with a flap or dark goggles.

(*e*) The only treatment I advise between the paintings is swabbing with lotion acid borac. 4th hourly and the application of ung. hyd. oxy. flav. 1 per cent.—thrice daily to the lid margins.

(*f*) Ice compresses in the more severe cases are advisable four hourly but they are by no means necessary in all cases.

What proportion of cases of keratitis are due to the virus of herpes simplex?

I found that in my recent paper of superficial punctate keratitis in Tasmania (1941) I did not give figures of the various types of herpetic infiltration of the cornea that I found, but I can give these now, and a comparative table detailing those found in the A.I.F. Middle East.

These figures are presented in Table I and II :—

TABLE I

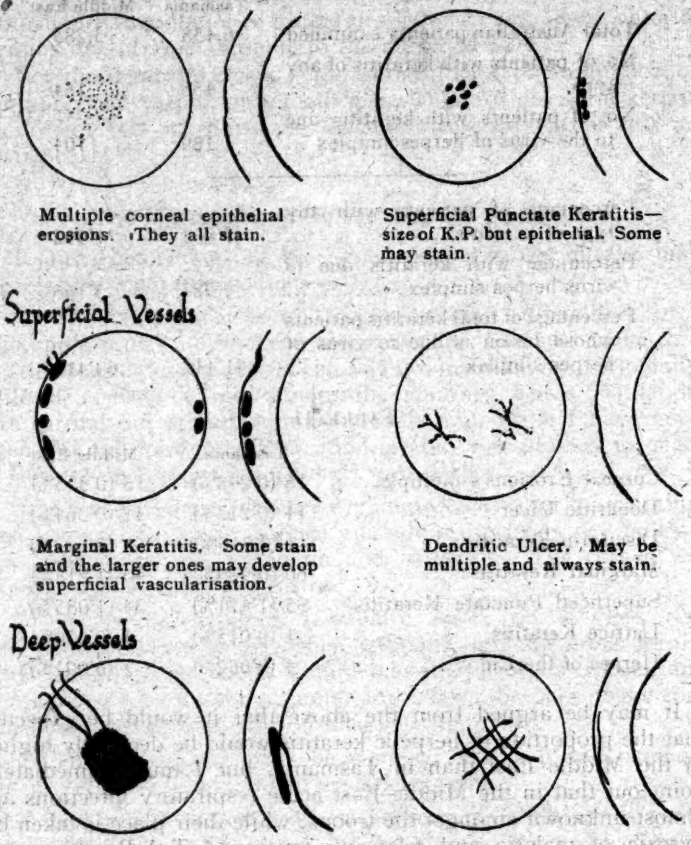
	Tasmania	Middle East
Total Australian patients examined	6,458	3,285
No. of patients with keratitis of any type	456	164
No. of patients with keratitis due to the virus of herpes simplex ...	189	104
Percentage of patients with any type of keratitis	7·063	4·992
Percentage with keratitis due to virus herpes simplex	2·927	3·166
Percentage of total keratitis patients whose lesion is due to virus of herpes simplex	41·447	63·415

TABLE II

	Tasmania	Middle East
Corneal Erosions—multiple...	16 (0·248%)	13 (0·395%)
Dendritic Ulcer	14 (0·217%)	12 (0·364%)
Disciform Keratitis	3 (0·046%)	4 (0·122%)
Marginal Keratitis	60 (0·929%)	40 (1·218%)
Superficial Punctate Keratitis	95 (1·471%)	34 (1·035%)
Lattice Keratitis	1 (0·015%)	1 (0·030%)
Herpes of the Lid	4 (0·062%)	3 (0·091%)

It may be argued from the above that it would be expected that the proportion of herpetic keratitis would be decidedly higher in the Middle East than in Tasmania, but I must immediately point out that in the Middle East acute respiratory infections are almost unknown amongst the troops, while their place is taken by pyrexia of malaria and relapsing fever and T.A.B. injections. Under these circumstances it is not surprising that the figures are so congruous. So we see that approximately 3 per cent. of eye disease, whether it be in Australians in Tasmania or the Middle East, are due to the virus of herpes simplex. But, owing to the fact that non-virus keratitis is decidedly less common in the Middle East than it is in Tasmania, we find that of the total cases of keratitis, 41·447 per cent. are due to this virus in Tasmania, while 63·415 per cent. are due to it in the Middle East. In brief, I find that in Australians in the Middle East no less than 2/3 of the keratitis met is of virus simplex aetiology, a fact, I feel, that is not yet realised.

FIG. 1



Multiple corneal epithelial erosions. They all stain.

Superficial Punctate Keratitis—size of K.P. but epithelial. Some may stain.

Marginal Keratitis. Some stain and the larger ones may develop superficial vascularisation.

Dendritic Ulcer. May be multiple and always stain.

Disciform Keratitis is parenchymatous and produces deep vascularisation.

Lattice Keratitis is subepithelial. It does not stain and is transient in form.

With regard to the individual figures detailed in Table II there is little comment, except that there is an increase in every type of virus keratitis in the Middle East, with the obvious exception of superficial punctate keratitis. I do not think that any conclusion can be deduced from this, other than that a totally different climate has slightly altered the proportions of the manifestation of an infectious disease. This indeed has been observed in almost every other infection.

Summary

(1) 63 per cent. of the keratitis in Australians in the Middle East is due to the virus of herpes simplex.

(2) The majority of these cases is due to a preceding true or artificial pyrexia or gross change of climate.

(3) Repeated paintings with $AgNO_3$ 2 per cent. are strongly indicated in marginal and superficial punctate keratitis.

(4) Carbolization is still the treatment par excellence for dendritic ulcer.

(5) Local heat or thermotherapy of any type is strongly contra-indicated in the treatment of any type of keratitis due to herpes simplex.

(6) Atropine is not necessary, except in disciform keratitis.

Conclusion

I must thank Colonel W. Summons, O.B.E., Commanding Officer of an Australian General Hospital, for permission to publish these figures, and I must acknowledge gratefully the assistance of the Sisters and Nursing Orderlies of this Hospital. Especially am I indebted to Sister B. Scott-Young, Sgt. K. MacFarlane and Private G. Petty, for their valuable and continuous help.

REFERENCES

DAVIS (1942).—*Arch. of Ophthal.*, February.

GIFFORD, S. (1942).—*Arch. of Ophthal.*, February.

GORDON, H. (1941).—*Proc. Roy. Soc. Med.*, Vol XXV, No. 1, p. 95.

HAMILTON, J. B. (1941).—*Brit. Jl. Ophthal.*, Vol. XXV, p. 1.

MACKIE and MCCARTNEY (1938).—Hand-book of Practical Bacteriology, Edinburgh.

ANNOTATION

Presence of Mind

The great Lexicographer, sometimes known as Surly Sam to differentiate him from Ben Jonson, defined presence of mind as readiness at need and quickness at expedients. It is a most useful asset, and we believe it to be a quality inborn in most people. Education may foster it but cannot produce it if it is not there. If an ophthalmic surgeon begins a cataract operation and finds, when he has completed his puncture and counter-puncture, that he has inserted the Graefe knife upside down, we believe the best thing to do is to withdraw the knife at once and postpone the operation for a day or two until the punctures have healed. Presence of mind will not as a rule enable one to rotate the knife through half a circle

without loss of aqueous. It is a state of affairs that indicates lack of presence of mind before beginning the operation, for this is the time to make sure that the edge of the knife is directed upwards. We do not say that such an event might not enable a nimble witted person to display presence of mind when he has to explain to the patient and to his relatives and friends why it was that the operation has had to be postponed, but we are not up to offering any suitable suggestions. In ophthalmological practice it is said that very occasionally the wrong eye has been excised for such a condition as choroidal sarcoma. Here no amount of presence of mind can conceal the fact that a terrible blunder has been made. We always adopted the plan of making sure which eye was affected and marking a cross on the forehead over that eye before the patient was taken to the threatre, with instruction to the nursing staff that the mark was not to be washed off. Care in the preliminaries in every case is the best guarantee that we shall not have to exhibit this inborn quality in such cases, even if it means that by lack of use, presence of mind may grow rusty.

A good example of presence of mind was exhibited on one occasion in the final examination for medicine. In the viva voce the candidate was told to examine the chest of a stout, middle-aged man. He found that the patient was completely dull to percussion, back and front, on one side, and no breath sounds were audible, while the heart was not displaced. When the examiner asked for a diagnosis the candidate was unable to offer one and said he didn't know. The examiner said brusquely "Why, hasn't he an empyema?" The candidate replied that no patient with an empyema capable of producing the signs found had any business to be an ambulant case at the examination hall, which was perfectly true and quite unanswerable. This answer did not adversely affect the result of the examination.

ABSTRACTS

MISCELLANEOUS

(1) **Loewenstein and Steel (Glasgow).—Retinal tuberous sclerosis (Bourneville's disease).** *Amer. Jl. Ophthal.*, Vol. XXIV, p. 731, 1941.

(1) **Loewenstein** and **Steel** describe the case of a boy aged 3½ years who had one eye excised for "pseudo-glioma" and subsequently suffered from epileptic fits but no sebaceous adenomata were present. Ophthalmoscopic examination of the remaining eye

showed grey-green tumours, exudative retinitis, retinitis proliferans and angiomatous nodules. Later this picture changed. The excised eye showed multiple tumours composed of nerve fibres, neural cells producing closed and open cysts, a thickened membrana limitans interna, pyramidal tumour of the optic disc, a choroidal angioma and an angiomatous growth on the surface of the retina. The authors state that the condition is a primary ectodermal malformation and the disturbance of balance of ectoderm and mesoderm leads secondarily to a mesodermal tumour.

H. B. STALLARD.

(2) **M'Glone, Joseph (Glasgow).—A case of chloroma.** *Glas. Med. Jl.*, August, 1942.

(2) **M'Glone** reports a case of chloroma in a boy of ten years, involving both orbits and the left maxilla, having probably originated in the sphenoid. The course was rapid, lasting 18 weeks from the onset of proptosis until severe haemorrhage from the naso-pharynx caused death.

The general post-mortem appearance of the tumour is well illustrated. Secondary deposits were found in the lateral sinuses, paravertebral tissues in the upper thorax, and in the heart; histologically these deposits were composed of round cell infiltrations and myelocytes. Green urine was present in the bladder but the colour faded on exposure. It re-appeared with the addition of sodium hydrosulphite and it is suggested that this may prove to be a useful test.

B. W. RYCROFT

(3) **St. Clair Roberts, B. H. (Worcester).—Remarks on some types of industrial cataract.** *Med. Press and Circ.*, November 4, 1942.

(3) To a special number of the *Medical Press and Circular* on industrial accidents, **St. Clair Roberts** contributes a very good account of some industrial cataracts. Having briefly dealt with traumatism, especially that due to perforation by metallic particles and the variety met with in country districts in those who pleach hedges, he mentions some of the chemical agents responsible; but the major part of his paper deals with physical agents and specially the type of cataract met with in chain makers, of which he is a recognized authority. The process of chain making is mostly localized in the Black Country still. Its details are set out and there is a good description of the type of cataract which occurs in the youngish middle-aged. The chain maker accepts the idea that it is customary to lose the sight in middle age. This, with the nature of their work, makes it very difficult to get them to use any form of protection in the shape of goggles. The largest chains are

made mostly by electrical machinery and the workers here are not so liable to suffer. The medium and small linked chains are still made by hand and here the workers are very likely to be affected. The author states that he has kept careful watch on a few cases who have been able to return to their work after removal of the cataract, and has never found any damage to occur at the macula.

Those of our readers who are interested in chain making might read F. Brett Young's novel "Far Forest," in which the background of the first part of the story is laid in one of the smithy hearths.

R. R. J.

(4) **Ballantyne, A. J. (Glasgow).—Recent work on vascular disease and its significance in medical ophthalmology.** *Glas. Med. Jl.*, July, 1942.

(4) In his William Mackenzie Memorial Address **Ballantyne** surveys recent work on vascular disease and its association with medical ophthalmology. He classifies the retinal vascular changes as follows:—

1. Calibre Changes.

An increase in diameter, although uncommon, is noticed in profound anaemias with a tendency to equality in the colour and calibre of the arteries and veins, whereas a general diffuse contraction is much more common and widely recognised in such conditions as advanced atrophy of the optic nerve.

2. Spastic Contraction of the Vessels.

The difficulty in distinguishing between a true spasm of the vessel wall and an organic contraction is pointed out. True spasm is to be found in eclampsia and Raynaud's disease. In 38 cases who complained of a temporary loss of vision, partial or complete, 19 of them appeared to be examples of transient angiospasm, either of retinal or cortical vessels. Moreover, vascular spasm may also be the cause of permanent loss of vision if it occurs in retinal vessels previously narrowed by sclerotic changes, and the estimation of prognosis is thereby rendered difficult. A case is quoted where symptoms suggested an arterial spasm in the left eye, in which vision failed to return; identical symptoms in the right eye completely recovered.

3. Angiospastic Retinopathy.

The significance of arterial and arteriolar spasm in hypertension is reviewed and the element of spasm is stressed. There is reason to believe that this commences first in vessels smaller than those normally visible to the ophthalmoscope, since gross spasm, as in

quinine poisoning, does not produce similar fundus lesions. However, it is suggested that the various fundus changes may be brought about by a difference in the circulating toxin.

4. Sheathing of the Retinal Vessels.

Sheathing of the retinal vessels shows two distinctions, namely, that of the intima, which causes irregular or patchy thickening, and that of the media, which is more uniform. Regressive changes of a hyaline or lipoid nature render the walls visible and produce the phenomenon of sheathing. Sheathing of the veins, on the other hand, may be due to active periphlebitis, as in Eales' disease, or to "cuffing" of the vessels as in leukaemia. It is shown that sheathing is due to many causes; the blood column disappears and is replaced by a white band, but it does not necessarily mean that the vessel is completely obliterated.

5. Arterio-Venous Crossings.

The precise cause of Marcus Gunn's sign is disputed. It is pointed out that compression of the vein alone is not responsible for the narrowing of the lumen for in true compression expansion of the vessel wall would be expected. Ballantyne believes that Gunn's phenomenon is due to veiling of the venous blood column as it recedes into the deeper layers of the retina and passes behind the artery.

6. New Vessel Formations.

These may be observed in four clinical forms:—

1. In hypertension with arteriosclerotic changes where intervals between principal retinal vessels are filled by a loose network of very fine venous branches which may, or may not, be traceable to their connections with the principal veins. This type is seen in its most complete form as a sequel of central retinal vein obstruction. Such venous collaterals go by the name of "rete mirabile." As a rule this network is intraretinal, but occasionally branches may perforate the internal limiting membrane of the retina and push forward into the vitreous cavity, giving type 2.

2. Although this formation may arise at practically any part of the fundus, the internal limiting membrane does, to some extent, offer a barrier to its forward growth, and probably for this reason most of these new vascular formations arise from the vessels on the disc where the limiting membrane is absent.

3. The third type is known as retinitis proliferans and is usually at a point where a retinal haemorrhage has broken through the internal limiting membrane. Organisation of the blood clot is responsible for the opaque white bands attached to the anterior surface of the retina, and there is a preponderance of mesoblastic tissue, much more solid and substantial than the mucoid framework

of the previous type, which is sometimes so tenuous that it scarcely causes any appreciable interference with vision. The explanation for this new vessel formation is said to be mechanical and purposive in the first instance, whilst the second type is extravagant and purposeless.

4. This type deals with new vessels closely related to the main trunks of the retinal veins. They are of large size and may be accompanied by formations of tufts, fans or networks of dilated capillaries, and practically always occur along with other gross pathological changes in the retinal veins. Excellent diagrams are added to amplify the description of these larger venous formations, and micro-photographs show their relationship to the layers of the retina. It is emphasised that these changes are more related to diabetes mellitus than to hypertension. Post-mortem appearances in a woman aged 61 years, suffering from diabetes, showed details of the rete mirabile in the retina and, in addition, a pre-retinal formation of new veins, which could be traced to a large retinal vein which had perforated the internal limiting membrane. It is probable that the network of vessels lay between two layers of a divided internal limiting membrane. The occurrence of the more unusual loops and networks of large-calibred vessels in ophthalmoscopic examination of so many diabetic cases convinced the author that he was dealing with a distinct clinical entity. He points out that hypertension is not essential, and that in many diabetes cases histological examinations have shown that the vessel changes in the retina fall more emphatically on the veins (phlebosclerosis). Also that abnormal fulness of the veins is one of the earliest retinal changes in some diabetics, and that the characteristic punctate haemorrhages are apparently derived from the post-capillary venous radicles; nor indeed, are these venous changes familiar in numerous cases of hypertension.

The pathogenesis of the venous plexus and calibre changes is not clear, but it is suggested that they may be due to a retardation of the blood stream in the central retinal vein. On the other hand, Foster Moore (1924) found only one diabetic in 61 cases of thrombosis of the central retinal vein, whereas in the author's group of 16 cases 15 were diabetic, and he holds the view that gross venous changes in the retinal vascular system justifies the diagnosis obtained in the majority of cases.

7. Oculo-Cerebral Syndromes.

These syndromes are represented by the von Hippel-Lindau disease, with one or more haemangiomatous tumours in the retina and associated with angiomatous cysts of the cerebellum and elsewhere. Angioma of the choroid, with associated lesions of the central nervous system, is known as the Sturge-Weber Syndrome.

Five cases of subarachnoid haemorrhage are described in detail, together with post-mortem findings. They ranged in age from 39 to 62 years. In the pathological details stress is laid on the distribution of haemorrhage in relation to the brain and the sheath of the optic nerves. Of these cases all had elevation of blood pressure, and in only one was the fundus free from haemorrhage. In four cases the source of the subarachnoid haemorrhage was discovered, and in three of these it was the rupture of an aneurysm of the circle of Willis. It was shown that haemorrhage could occur not only in the dural sheath, the subdural and subarachnoid space, but also in the optic nerve and pial sheath, in the sclera near the porus opticus, in the capsule of Tenon, among the orbital fat and muscles, in the fibrous sheaths surrounding the ciliary ganglion, in the pial covering of the chiasma, and in the chiasma itself. The old explanation that blood, effused at the base of the brain, passed along the optic nerve sheaths through the optic canal is considered to be not entirely satisfactory, since there is not necessarily continuity between these various extravasations of blood.

The author is of opinion that the intra-cranial and intra-orbital haemorrhages are independent, and so also are those haemorrhages within the optic nerve sheaths and in the retina. He submits the view that the precipitating cause of these haemorrhages is the sudden rise of intra-cranial pressure which occurs at the moment of rupture of the aneurysm acting directly on the venous sinuses and causing extensive stasis and resulting extravasations.

A short bibliography completes the address.

B. W. RYCROFT.

(5) **King, A. and Walsh, F. B. (Baltimore).—Temporary amaurosis and hemianopsia due to epilepsy.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 398, 1942.

(5) **King** and **Walsh** describe two cases in which visual defects persisted after an epileptic attack when other functions had returned fully. One was a female child, aged 22 months, in whom blindness of both eyes persisted for 10 days after an epileptic seizure. During this period she showed no blink reflex to objects held close to her eyes and there was no evidence of nystagmus on looking down at a revolving drum. These became apparent on the tenth day. The authors comment that Ashby and Stephenson (*Lancet*, Vol. I, pp. 1294 and 1616, 1903), recorded one case of persistent blindness after an epileptic attack.

The second case was a male negro, aged 45, who had a post-epileptic left homonymous hemianopia which lasted a month and then recovered. In the blind part of the field he had visual hallucinations. In the discussion of these cases the authors support the conjecture that in cases where the pupillary reflexes are intact and

there is homonymous hemianopia there is temporary exhaustion of the visual cortex associated with the epileptic discharge.

H. B. STALLARD.

(6) **Dunnington, John H., and Wheeler, Maynard C. (New York).—Operative results in two hundred and eleven cases of convergent strabismus.** *Arch. of Ophthal.*, July, 1942.

(6) This paper by **Dunnington** and **Wheeler,** although covering familiar ground, contains many points of interest. The present position of squint surgery is put rather neatly by them in the aphorism that "the mathematical millenium is not in sight," and the results they quote go to prove this. In analysing the cases it was found that in as many as 28 per cent. the strabismus was present from birth though the average age of onset was 1·9 years. Amblyopia in one eye was present in roughly 50 per cent. of cases, and in 31 where constant occlusion was efficiently carried out, vision of 20/50 or better was obtained in 70 per cent. With regard to refractive error it is remarkable that only 17 per cent. of cases showed over 4D. of hypermetropia and that 68 per cent. showed less than 3D. No relationship was found to exist between the amount of deviation and the refractive error. In 71 per cent. there was a deviation of 20° or more, the average being 23°.

Estimation of the post-operative result was difficult and the authors took as an arbitrary definition of a cure a residual deviation of not more than 10 prism dioptres for either distance or near vision. Using this, they found that one operation was sufficient to effect a cure in 43 per cent. of cases. The under corrections varied in severity, but in most of them approximated to 20 prism dioptres. It is notable that abnormal retinal correspondence was more frequent in the failures than in the cures. The figures give little guidance as to the best age at which to operate, except for the observation that the highest proportion of over-corrections (20 per cent.) was obtained in the age group 3-8 years. The results obtained in alternating squint were inferior to those in the uniocular variety, the tendency being towards under-correction in the former and over-correction in the latter. Although the effect of the same operation varied from case to case, it was found that the average correction obtained by recession on one eye only was 7·3 prism dioptres, while that obtained by a 4mm. recession combined with a 10mm. resection was 40 prism dioptres. The authors rightly remark, however, that much depends on the technique employed at operation.

Post-operative diplopia was frequent as a transient complaint, but in only three instances did it show any tendency to be permanent and in only one of these was it distressing.

F. A. W-N.

CORRESPONDENCE

CHOROIDAL MELANOMA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Mr. Foster Moore has very kindly shown us his letter (p. 46). The question of a congenital patch in the pigment epithelium was considered; but we had not thought such a patch could enlarge. The letter relieves my mind very much. Pritchard and I got to know and like the patient, and we had an unpleasantly guilty feeling that perhaps we were failing in our duty in not removing his eye. After Mr. Foster Moore's letter we shall watch the case with diminished apprehension.

Yours sincerely,

L. H. SAVIN.

LONDON,
December 16, 1942.

UNUSUAL CASES OF CONJUNCTIVITIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I am grateful to Dr. J. Ruszkowski for pointing out the obvious omission of "artefact" in the differential diagnosis of the four unusual cases of conjunctivitis which I recently described in the *Brit. Jl. of Ophthal.*, Vol. XXVI, p. 467. The possibility of a self-inflicted condition was, in fact, considered in each of these cases and was regarded as improbable.

As Dr. Ruszkowski notes, the localisation to one eye and the marked involvement of the lower fornix are highly suggestive. On the other hand the injection was not confined to this site but was also present beneath the upper lid and in the region of the canthi. Again, the superficial keratitis in two of the cases was of a type commonly associated with prolonged conjunctivitis, while in the two other cases the corneae remained unaffected throughout; in neither were there at any time the multiple grey opacities in the corneal epithelium which I have noted, particularly in the lower half of the cornea, in several cases where the diagnosis of a self-inflicted condition seemed reasonably sure.

I had each of these men under my care as an in-patient for many weeks. At no time was there any indication of trauma to the skin of the lids and it seemed difficult to suppose that such severe reaction could have been maintained without some such result. It is possible, of course, that soap or some other irritant was introduced

into the conjunctival sac, but of this, again, no suggestive evidence was forthcoming. Finally, each man happened to be of a particularly straightforward and direct type and I found it difficult to associate a self-inflicted disability with either.

I am, etc.,

E. F. KING,
Major, R.A.M.C.

December, 1942.

NOTES

Death

WE regret to record the death of William Herbert Lowry, of Toronto, on July 13, 1942. An obituary notice appeared in the October number of the *Canadian Medical Association Journal* and from it the following facts are abstracted with the usual acknowledgements.

Born in 1880, the son of a doctor, he graduated M.D. at Toronto in 1901. Almost immediately he decided to practise ophthalmology, and after post-graduate study in London and Birmingham he was appointed to the staff of the Hospital for Sick Children, where, in 1919, he became chief ophthalmic surgeon. Ten years later he succeeded MacCallum as Professor of Ophthalmology at the University of Toronto. Resigning the post in 1941 through ill-health he was elected Emeritus Professor. He served in the last Great War in England and Salonika and rose to the rank of Lieutenant-Colonel. Lowry was a good sportsman, a fine shot, and he played golf and tennis well. "In the operating room he was deft and sure, with a conservative but hopeful outlook which meant much to his patients who held him in high esteem." He was also an earnest and painstaking teacher, particularly interested in undergraduate training.

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Royal Society of Medicine

THE meetings of the Section of Ophthalmology during the session 1942-43 will be held as follows:—

Friday, March 12, at 2.30 p.m.
Friday, April 30, at 5.0 p.m.
Friday, June 11, at 5.0 p.m.

Cases and specimens will be shown half-an-hour before the advertised times of the meetings.

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Special Notice

WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.